Report No. NADC-91079-60

THE SCOPE OF ACCELERATION - INDUCED LOSS OF CONSCIOUSNESS RESEARCH

James E. Whinnery, Ph.D., M.D.
Air Vehicle and Crew Systems Technology Department (Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

4 JULY 1991

FINAL REPORT
Period Covering May 1988 to May 1991

Approved for Public Release; Distribution is Unlimited

Prepared for
Air Vehicle and Crew Systems Technology Department (Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

NOTICES

REPORT NUMBERING SYSTEM — The numbering of technical project reports issued by the Naval Air Development Center is arranged for specific identification purposes. Each number consists of the Center acronym, the calendar year in which the number was assigned, the sequence number of the report within the specific calendar year, and the official 2-digit correspondence code of the Command Officer or the Functional Department responsible for the report. For example: Report No. NADC-88020-60 indicates the twentieth Center report for the year 1988 and prepared by the Air Vehicle and Crew Systems Technology Department. The numerical codes are as follows:

CODE	OFFICE OR DEPARTMENT
00	Commander, Naval Air Development Center
01	Technical Director, Naval Air Development Center
05	Computer Department
10	AntiSubmarine Warfare Systems Department
20	Tactical Air Systems Department
30	Warfare Systems Analysis Department
40	Communication Navigation Technology Department
50	Mission Avionics Technology Department
60	Air Vehicle & Crew Systems Technology Department
70	Systems & Software Technology Department
80	Engineering Support Group
90	Test & Evaluation Group

PRODUCT ENDORSEMENT — The discussion or instructions concerning commercial products herein do not constitute an endorsement by the Government nor do they convey or imply the license or right to use such products.

Reviewed By: N/A Date: _____
Branch Head

Reviewed By: _____ Date: 2 Nov 91
Division Head

Reviewed By: _____ Date: 2 Nov 91
Director/Deputy Director

REPORT DOCUMENTATION PAGE

Form Approved
OMB No 0704-0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	4 July 1991	Final 5/89 - 5/91

4. TITLE AND SUBTITLE
THE SCOPE OF ACCELERATION - INDUCED LOSS OF CONSCIOUSNESS RESEARCH

5. FUNDING NUMBERS

6. AUTHOR(S)
James E. Whinnery, Ph.D., M.D.

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Air Vehicle and Crew Systems Technology Department
(Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

8. PERFORMING ORGANIZATION REPORT NUMBER
NADC-91079-60

9. SPONSORING MONITORING AGENCY NAME(S) AND ADDRESS(ES)
Air Vehicle and Crew Systems Technology Department
(Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

10. SPONSORING MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION AVAILABILITY STATEMENT
Approved for Public Release; Distribution is Unlimited

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

Acceleration (+G_z) induced loss of consciousness (G-LOC) is a major fighter aviation medicine problem. To effect an ultimate solution for G-LOC it is critical to have a thorough understanding of the problem. Research related to unconsciousness is spread across a multitude of disciplines. It is therefore a valuable exercise to develop the overall scope of relevant research that pertains to G-LOC. This not only serves to bring together the body of information that is important for understanding G-LOC, it also provides for the development of the scope of importance that G-LOC research has for other scientific and clinical disciplines. The purpose of this review is to establish the relationship between biomedical research whose primary goal is aviation oriented and the clinical and basic research that is intricately associated with unconsciousness and may benefit from the results of G-LOC research.

14. SUBJECT TERMS
ACCELERATION
LOSS OF CONSCIOUSNESS
UNCONSCIOUSNESS
SYNCOPE
FIGHTER AVIATION
NEUROPHYSIOLOGY
CARDIOLOGY

15. NUMBER OF PAGES

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
UNCLASSIFIED	UNCLASSIFIED	UNCLASSIFIED	SAR

CONTENTS

FIGURES

TABLES

THE SCOPE OF ACCELERATION-INDUCED LOSS OF CONSCIOUSNESS RESEARCH

The importance of establishing the scope of acceleration ($+G_z$) induced loss of consciousness (G-LOC) research became very apparent early in the efforts to understand the phenomenon. One of the first tasks a researcher has when attacking a problem is to review as much of the existing literature on the topic as possible. Appropriately, the initial literature of importance is the body of aviation related material concerning loss of consciousness in the aerospace environment. Just to enter this environment represents a life-threatening event, requiring life support and protection systems to prevent loss of consciousness and subsequent injury and/or death. Much of the aviation literature concerning loss of consciousness is operationally applied or clinically oriented. Fundamental research concerning the neurophysiological aspects of loss of consciousness in the aviation literature is not very extensive, therefore, review of a much wider spectrum of research literature is required.

For the fighter aviation medicine specialist, such a literature review might begin by searching for consciousness, unconsciousness, loss of consciousness, altered consciousness, fainting, and/or syncope. With the exception of fainting and syncope they will find very little quantitative data of specific usefulness. Traditional clinical medicine preceded aviation medicine and therefore the roots of understanding unconsciousness should be buried in clinical medicine. Loss of consciousness, syncope, or fainting however, is considered only a symptom in clinical medicine. This continues to be true even today in formal medical education. Indeed this is true. In clinical medicine syncope occurs when an underlying problem such as a cardiac dysrhythmia exists. The medical goal in this particular case is oriented toward correcting the primary cardiac problem. This goal will solve the syncopal problem since it was only a symptom of the underlying primary cardiac problem. The same is true for primary neurologic problems that result in loss of consciousness. Most of the articles with syncope in the title are devoted toward the underlying abnormality that leads to conditions that produce the neurologic alterations that are *the cause of syncope*. Significant effort has been devoted toward developing a qualitative description of syncope. This is done in an effort to try and identify the underlying primary cause of the symptom, especially differentiation of epilepsy (or other neurologic pathology) from cardiovascular and other systemic causes.

Detailed qualitative description of the syncopal episode is usually minimal. Quantitative and kinetic descriptions are almost non-existent. This is probably due to the fact that syncopal episodes are infrequently observed and occur sporadically. Indeed it is likely that a large number of medical professionals, including specialists who effectively diagnose and treat the pathophysiologic problems underlying syncope in clinical patients, may never or only rarely have seen a syncopal episode. Medical education would therefore be well served by utilizing the videotape evidence which vividly illustrates the characteristics of the G-LOC syndrome in completely normal humans. Understanding the response of the normal CNS to ischemia/hypoxia should be of value when making diagnostic decisions. Description of syncopal characteristics frequently comes from the patient or a non-medical observer. Even when a clinical specialist does have the good fortune of observing a loss of consciousness episode, they usually do not have a stopwatch and are not poised to make kinetic measurements in the midst of the syncopal episode. Since a multitude of things (the G-LOC syndrome) are happening over a very short period of time, without video recording of the episode, accurate kinetic measurements associated with a loss of consciousness episode are virtually impossible. The potential for a life-threatening etiology underlying the loss of consciousness presses the medical professional to very appropriately be in a treatment and intervention mode in such a situation. The potential life-threatening and injurious situation associated with a loss of consciousness episode in patient populations is probably why so much concern has persisted to date concerning the induction of loss of consciousness even in healthy individuals. Rather than logically evaluating the more than adequate resistance of the bodily tissues to the very transient ischemia/hypoxia, associated with many loss of consciousness episodes (including G-LOC), the anxiety and timidity associated with inducing loss of consciousness remains tied to the underlying clinical problems which lead to the episode and not the loss of consciousness itself. Overall

it is not surprising, therefore, that establishment of the quantitative kinetic analysis of loss of consciousness has been reserved to be the domain of the modern fighter aviation medicine specialist.

Indeed, G-LOC is a symptom associated with exceeding tolerance to $+G_z$-stress. However, it very appropriately becomes a problem when aircraft and aircrew are lost as a result of this neurologic symptom. G-LOC must be considered a primary fighter aviation medicine problem. Only in this way can it gain the prominence necessary to allow efforts to be directed toward a solution. The history of aviation medicine aptly illustrates that the lack of such recognition by early researchers resulted in little effort aimed directly at G-LOC. Hence, it has remained a problem, one that costs the nation lives and exceptionally costly weapon systems. Elevating G-LOC to a problem status has increased the attention given to acceleration medicine and physiology. A solution, however, remains elusive. It is evident that uninspired acceleration technicians who consider it not worth investigating the neurophysiological cause and mechanism of G-LOC, have lost the basic scientific zeal, inquisitiveness, and search for truth that may benefit a wide spectrum of human research that is contained within the scope of acceleration-induced G-LOC (9).

With loss of consciousness traditionally being considered only a symptom, we have a paucity of clinical information devoted to understanding the loss of consciousness phenomenon. The description of syncope, even in the most up-to-date clinical text books, remain grossly lacking in qualitative and quantitative detail. In fighter aviation medicine we are left with a historic data base composed of some qualitative information from clinical medicine and minimal incomplete qualitative and quantitative data from aviation medicine.

The study of Rossen, Kabat, Anderson published in 1946 stands as a landmark study (5). It is indeed a study aimed at understanding loss of consciousness in humans induced by strangulation. The investigators developed, for the first time, some of the techniques to quantitatively measure loss of consciousness, something which remained to be described at all by acceleration physiologists. A full quantitation of all the symptoms, however, remained lacking even in the Rossen, Kabat, Anderson study. The very fine study, therefore, remains limited in both scope and its statistical analysis of the data (4). Only 28 data points for recovery of consciousness were given in the study. Minimal similar research has been reported over the subsequent 46 years.

Once the researchers discover the lack of information gained by searching for syncope, fainting, and loss of consciousness, they may generally turn their search toward specific underlying clinical causes such as anoxia, hypoxia, hanging, and strangulation. This is of some help, but again little if any kinetic information or detailed descriptions will likely be uncovered. The major importance of such studies lies in the description of the resulting central nervous system (CNS) pathology, lesion-symptom correlation, lower limit of insult duration and magnitude necessary to produce pathology, regional CNS area ischemia/hypoxia sensitivities, and the effects of combined anoxic/ischemic insults. The fact that these reports are generally clinical in nature (patients) indicates that some residual problem or at least the need for medical intervention is present. This means that the duration and magnitude of the insult are considerably greater than that necessary to induce simple unconsciousness in healthy individuals. G-LOC results from altered function not altered integrity of the CNS. It is for this reason that the next approach to find information about loss of consciousness is likely to search for research reports involving "acute" reduction of blood flow to the CNS. To the dismay of the aviation researcher, they will find "acute" usually means 3 to 10 minutes of CNS ischemia to the majority of clinically oriented investigators. The focus of such research is related to altered CNS integrity such as strokes, cerebrovascular accidents, and atherosclerotic cerebrovascular disease. This is undoubtedly driven by the funding of research that promises to solve specific clinical problems. The vast majority of this basic research involves animals also. Although animal research is very important, it is not of specific applicability to human unconsciousness since it is difficult to know exactly when and whether the animal is or is not conscious and the existence or absence of psychophysiologic symptoms. Invasive

experimental investigation, however, can generally only be accomplished with animals (and human patients). Since the very highest levels of cortical activity are involved in consciousness, humans are eventually required for an ultimate understanding of G-LOC. Since we are interested in G-LOC in fighter aircrew, healthy humans are optimum for evaluation of G-LOC phenomena.

The next literature area to provide some information on human unconsciousness could be reports involving sustained cardiac dysrhythmias (ventricular fibrillation, asystole) in human patients. Some work prior to the advent of cardiac pacemakers and defibrillation is of interest; however, this is usually reported only for individuals with advanced cardiovascular and/or cerebrovascular (neurovascular) disease. Ancillary information of importance includes the neurosurgical and pathological reports regarding states of consciousness resulting from disease or trauma.

I have previously emphasized the complex nature of the reaction of the body to CNS ischemia/hypoxia by defining the G-LOC syndrome (6). With the recognition of symptoms such as myoclonic convulsions and memorable dreams that occur in association with G-LOC (the unconsciousness), additional areas of clinical medicine and basic research become of interest. Valuable insight can be gained from research involving seizures, convulsive activity, sleep, dreaming, memory, and learning. Understanding the neurologic mechanisms underlying these phenomena and relating them to the G-LOC syndrome is extremely fruitful. As a specific example that such similarities exist, Table I illustrates the almost identical symptoms associated with narcolepsy and the G-LOC syndrome (1). Since much is known concerning their mechanisms, perhaps these areas are the most valuable of all the currently available information. With similar symptoms reflecting similar neurologic substrates and mechanisms, much use can be made of these studies. Clinical medicine is interested in understanding such events as seizures, since they are primarily neurologic problems. Unconsciousness, as occurs in fighter aviation, should be treated in a similar fashion. It is a primary dysfunction of the CNS resulting *from ischemia/hypoxia. Dysfunction* of the CNS may be incorrect terminology since G-LOC is a normal process of the CNS responding to ischemic/hypoxic insult. Considering G-LOC as a cardiovascular disorder fails to progress to the ultimate neurophysiological cause of G-LOC.

Actual studies involving induction of loss of consciousness in healthy humans are extremely few in number. Besides the 1946 studies of Rossen, Kabat, and Anderson, unconsciousness was induced by Duvoisin using physiologic (Valsalva's and Weber's) maneuvers. Duvoisin's excellent studies are extremely valuable, specifically with respect to the documentation of the completely safe conduct of such studies (2,3). They are similar to the early studies in aviation medicine and acceleration physiology which provide only qualitative information. Very little kinetic information is presented in these studies or the early acceleration studies. Even where unconsciousness frequently occurs, it is usually not recorded for kinetic analysis. Information does exist concerning the frequency of loss of consciousness relative to blood donation. The frequency of occurrence is indeed high and without sequelae; however, the kinetics and characteristics are not usually given in detail.

A wide range of literature therefore exists that is important for the fighter aviation medical scientist to become familiar with. The general categorization of pertinent literature is given in Table II. Despite this wide range of data, in the final analysis, there exists almost no kinetic information describing loss of consciousness, associated symptoms, and subsequent recovery. The task in the end remains for fighter aviation medical scientists to carefully define the detailed characteristics, kinetics, and mechanism of G-LOC. Even though the existing literature fails to provide many of the answers one would hope for, it does provide solid information relative to the lack of adverse events associated with transient unconsciousness similar to that which occurs with G-LOC. Such studies can be safely accomplished without lasting sequelae in completely healthy humans (7,8).

TABLE I. SYMPTOMS COMPARISON OF NARCOLEPSY AND G-LOC

NARCOLEPSY	G-LOC
VISUAL SYMPTOMS	VISUAL SYMPTOMS
CATAPLEXY (paralysis except respiratory muscles)	LOSS OF MOTOR FUNCTION (paralysis except respiratory muscles)
SLEEP	LOSS OF CONSCIOUSNESS
AMNESIA	AMNESIA (for the event)
MEMORY PROBLEMS	MEMORY PROBLEMS
HALLUCINATIONS	DREAMLETS
AUTOMATIC BEHAVIOR	MYOCLONIC CONVULSIONS

TABLE II. PERTINENT RESEARCH ON UNCONSCIOUSNESS

1. HUMAN NEUROSURGICAL RESEARCH
2. ANIMAL NEUROLOGICAL RESEARCH
3. HUMAN NEUROLOGICAL RESEARCH
4. HUMAN NEUROPATHOLOGY
5. CARDIOVASCULAR RESEARCH
    a. DYSRHYTHMIAS
    b. MYOCARDIAL INFARCTION
    c. SUDDEN (NEAR) DEATH
    d. CARDIOPULMONARY RESUSCITATION
    e. SYNCOPE
6. PHYSIOLOGIC ALTERATIONS
    a. HYPOXIA/ANOXIA/ALTITUDE STRESS
    b. LOWER BODY NEGATIVE PRESSURE/HYPOTENSION
    c. TILT TESTING
    d. VAGAL MANEUVERS
    e. TRAUMA (CONCUSSION)
    f. HYPOTHERMIA
    g. BREATH HOLDING/DIVING
7. PHARMACOLOGIC ALTERATIONS
    a. ANESTHESIA
    b. SEDATIVE-HYPNOTICS
    c. HYPOGLYCEMICS (DIABETES)
8. SEIZURE DISORDERS
    a. GRAND MAL
    b. PETIT MAL
    c. NARCOLEPSY (CATAPLEXY)
9. SLEEP
10. DREAMING
11. MEMORY
12. LEARNING
13. VISION
14. EVOLUTIONARY BIOLOGY
15. NEAR DEATH PHENOMENON

Based on the scientific and medical disciplines that contain research of importance for G-LOC, it is possible to develop a list of research areas that should benefit from the results gained from G-LOC research. As shown in Table III, the scope of benefits gained from G-LOC is very wide. It includes clinical and aviation medicine, basic neuro- and cardiovascular physiology, evolutionary biology, and even near-death phenomena. The qualitative description of the G-LOC syndrome resulting from specific types of ischemic/hypoxic insults to the CNS are of central significance to many pathophysiological processes. Refining the qualitative description, making it a quantitative description, and defining the kinetics of the processes are key to the delineation of the mechanism of unconsciousness. The mechanism of maintaining conscious function, its loss, and its recovery are important for understanding the very basis of cardiovascular and neurologic control within the body. The need for consolidating all pertinent research is evident if maximum progress toward solving G-LOC is to expeditiously proceed.

If indeed the current theory of G-LOC is validated, the entire heart-brain axis control mechanism may become integrated and unified. Although existing neurophysiologic terminology refers to neural control as activation and inhibition and cardiovascular terminology refers to cardiac control as sympathetic and parasympathetic, they may actually be one in the same. Neural control balance is key to the normal function of both the heart and the brain. As illustrated in Figure 1, both cardiac and brain function are maintained by the delicate balance of inhibition and activation integrated within the reticular formation and cardio-respiratory centers of the brain stem.

The study of acceleration physiology is the study of our evolutional adaptation to the gravitational environment as upright creatures. Many of the disease processes which affect us are related to the adverse impact of gravitation. At the end of our lives, the terminal loss of neurologic function represents death. Death with its loss of CNS tissue integrity must be preceded by loss of CNS function. The loss of CNS function is loss of consciousness, and as such, is the closest glimpse we mortals can get to understanding death. The study of acceleration and G-LOC therefore covers the entire spectrum of our being and represents a core unification of the study of human life.

TABLE III. THE SCOPE OF G-LOC RESEARCH

A. CLINICAL MEDICINE
   1. CARDIOPULMONARY RESUSCITATION
   2. CARDIOPULMONARY BYPASS TECHNIQUES
   3. CARDIAC DYSRHYTHMIAS
   4. CARDIAC VALVULAR DISEASE
   5. CEREBROVASCULAR DISEASE AND ACCIDENTS
   6. SYNCOPAL DISORDERS

B. AVIATION AND SPACE MEDICINE
   1. PERFORMANCE ENHANCEMENT
   2. SAFETY OF FLIGHT
   3. MISSION ENVELOPE EXPANSION
   4. MISHAP INVESTIGATION
   5. OCCUPATIONAL MEDICINE

C. BASIC NEUROPHYSIOLOGY
   1. GLOBAL CNS RESISTANCE TO ISCHEMIA/HYPOXIA
   2. CNS SUBSYSTEM FUNCTIONAL TOLERANCE TO ISCHEMIA/HYPOXIA
   3. CNS CIRCUITRY NECESSARY FOR MAINTAINING CONSCIOUSNESS
   4. CONVULSIVE PHENOMENON
   5. DREAMING
   6. SLEEP
   7. MEMORY
   8. LEARNING
   9. CNS CELLULAR ENERGY STORES
   10. BIOCHEMICAL REACTION KINETICS SUPPORTING CONSCIOUSNESS
   11. INSIGHT INTO THE HUMAN EVOLUTIONARY PATHWAY

D. BASIC CARDIOVASCULAR PHYSIOLOGY
   1. BRAIN STEM CONTROL INTEGRATION
   2. AUTONOMIC NERVOUS SYSTEM BALANCE
   3. CARDIOVASCULAR RESERVE AND REFLEXES

E. OTHER
   1. ANESTHESIA
   2. DIABETES
   3. CNS DISEASE/TRAUMA
   4. EVOLUTIONARY BIOLOGY
   5. NEAR DEATH PHENOMENA/TERMINALLY-ILL PATIENTS
   6. SPORTS MEDICINE

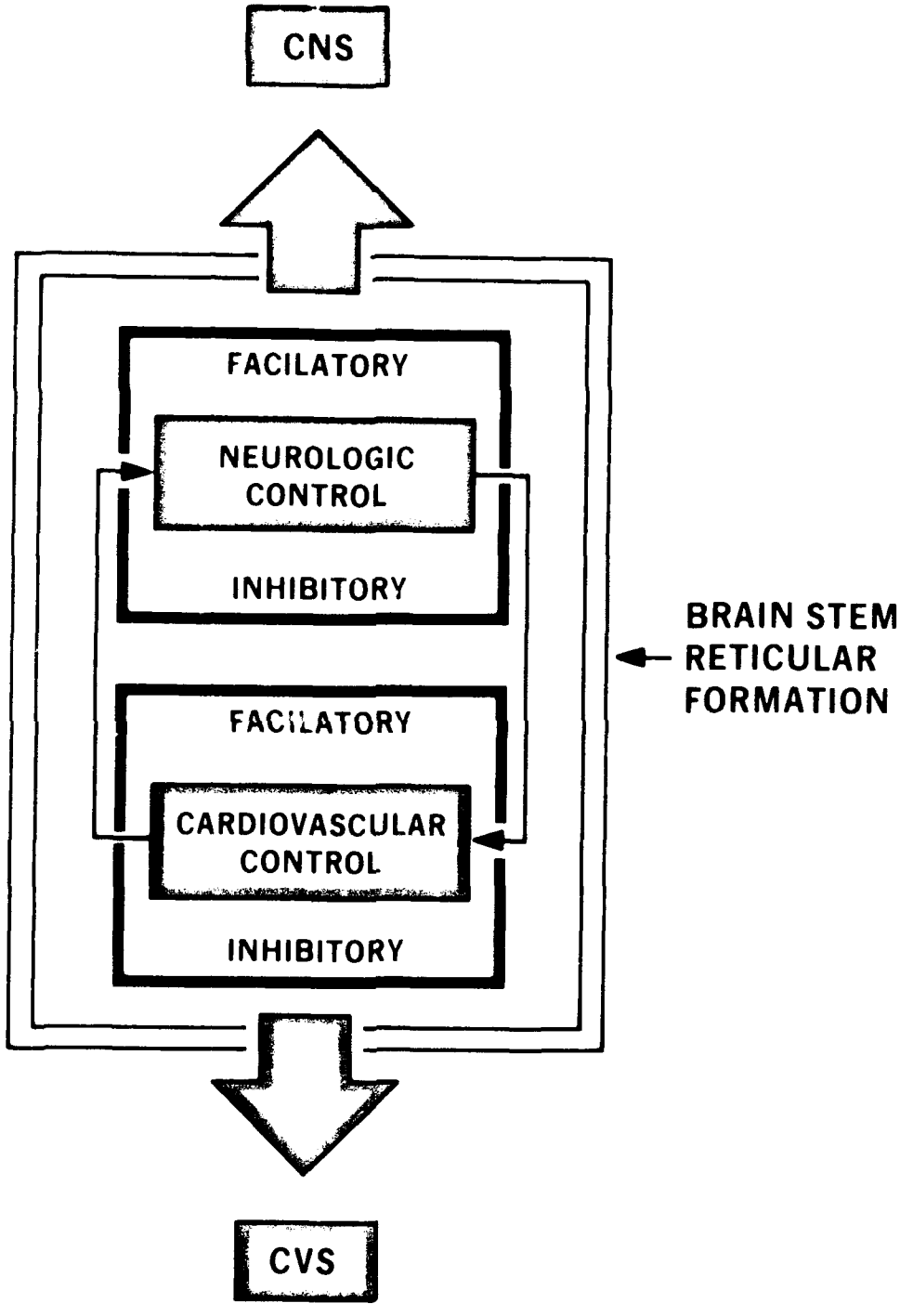

Figure 1. Theoretical Mechanism Involving Integration Of Control Of The Central Nervous System (CNS) And Cardiovascular System (CVS) Within The Brain Stem Reticular Formation.

REFERENCES

1. Aldrich M.S. Narcolepsy, N.E.J.M. 323: 389-394; 1990.

2. Duvoisin R.C. Convulsive syncope induced by the Weber maneuver. Arch. Neurol. 7:219-226; 1962.

3. Duvoisin R.C., Kruse F., Saunders D. Convulsive syncope induced by the Valsalva maneuver in subjects exhibiting low G-tolerance. Aerosp. Med. 33:92-96, 1962.

4. Forster E.M., Whinnery J.E. Statistical analysis of the human strangulation experiments. (Aviat. Space Environ. Med. submitted).

5. Rossen R., Kabat H., Anderson J. Acute arrest of cerebral circulation in man. Arch. Neurol. Psych. 50:510-528; 1943.

6. Whinnery J.E. The G-LOC syndrome. Naval Air Development Center Technical Report No. NADC-91042-60, 31 October 1990; Warminster, Pennsylvania 18974-5000.

7. Whinnery J.E. Defining risk in aeromedical unconsciousness research. Aviat. Space Environ. Med. 60:688-694; 1989.

8. Whinnery J.E. Medical considerations for human exposure to acceleration-induced loss of consciousness. Aviat. Space Environ. Med. 62:618-623; 1991.

9. Wood E.H. The state of G-LOC. IEEE Engineering in Medicine and Biology. (in press)

DISTRIBUTION LIST (Continued)
Report No. NADC-91079-60

	No. of Copies
Air Force Office of Scientific Research Bolling AFB, DC 20332-6448	1
Naval Aerospace Medical Institute Naval Air Station, Pensacola, FL 32508	2
FAA Civil Aeromedical Institute Oklahoma City, OK 73124	1
NASA Ames Research Center Moffott Field, CA 94035	1
NASA Johnson Space Center Houston, TX 77058	1
Dr. Dov Jaron Drexel University Philadelphia, PA 19104	1
Dr. Steven Dubin, Drexel University Philadelphia, PA 19104	1
Commandant of the Marines Headquarters Marine Corps Washington, DC 20380	1
Naval Weapons Center China Lake, CA 93555-6001	1
Naval Air Force U.S. Pacific Fleet NAS North Island San Diego, CA 92135	1
Commander Naval Air Force, U.S. Atlantic Fleet Norfolk, VA 23511-5188	1
Center for Naval Analyses 4401 Fort Ave., P.O. Box 16268 Alexandria, VA 22302-0268	1
U.S. Air Forces In Europe (USAFE) Office of the Command Surgeon/SG APO, New York 09094-5001	1
Mr. Robert Montgomery Aeromedical Training Institute ETC Corporation Southampton, PA 18966	1

DISTRIBUTION LIST (Continued)
Report No. NADC-91079-60

No. of Copies

Dr. David Glaister, Royal Air Force 1
Institute of Aviation Medicine
Farnborough, Hants; United Kingdom

Mr. Michael Paul, Defense and Civil Institute 1
of Environmental Medicine
1133 Sheppard Avenue
P.O. Box 2000
Downsview, Ontario, Canada

Mr. Floyd McGowan 1
P.O. Box 35
Chapman, Alabama 36015

Air University Library 1
AUL/LSE, Maxwell AFB, AL 36112

USAFSAM 1
Brooks AFB, TX 78235
(Strughold Aeromedical Library/TSK-4)

Commander, USAF Armstrong Aerospace 1
Medical Research Lab
Wright-Patterson AFB, OH 45433

Commander, Naval Training Systems Command 1
12350 Research Parkway
Orlando, FL 32826

U.S. Air Force Tactical Air Command 1
Office of the Command Surgeon (SGP)
Langley AFB, VA 23665

Naval Air Development Center 32
Warminster, PA 18974-5000
(30 Copies for Office of Chief Aeromedical Scientist)
(2 Copies for Code 8131)

Commander, Naval Air Systems Command 8
Washington, DC 20361
(3 Copies for AIR-320R)
(2 Copies for AIR-931H)
(1 Copy for AIR-531B)
(2 Copies for AIR-5004)

1299th Physiological Training Flight 1
Malcolm Grow USAF Medical Center
Andrews AFB, Washington, DC 20331-5300

DISTRIBUTION LIST
Report No. NADC-91079-60

	No. of Copies
Defense Technical Information Center Bldg. 5, Cameron Station, Alexandria, VA 22314	2
Chief of Naval Operations Dept of the Navy, Washington, DC 20350 (1 Copy for NOP-506N) (1 Copy for NOP-591) (1 Copy for NOP-05H) (1 Copy for NOP-09E)	4
Naval Aerospace Medicine Institute Naval Air Station, Pensacola, FL 32508	2
Office of Naval Research Code 1433, Washington, DC 20375 (1 Copy for Code 1433)	1
Naval Postgraduate School Monterey, CA 93940	1
USAF School of Aerospace Medicine USAFSAM/VNB, Brooks AFB, TX 78235 (1 Copy for Dr. Kent K. Gillingham)	1
USAF School of Aerospace Medicine Office of the Chief Scientist, Brooks AFB, TX 78235 (1 Copy for Dr. Russell R. Burton)	1
USAF School of Aerospace Medicine USAFSAM/VNB, Brooks AFB, TX 78235 (1 Copy for Dr. John W. Burns)	1
Air National Guard Support Center Mail Stop #18, Andrews AFB, DC 20331 (1 Copy for ANGSC/SG)	1
Advanced Physiologic Training Unit 833 Med Gp/SGT, Holloman AFB, NM 88330 (1 Copy for 833 Med Gp/SGT)	1
Federal Aviation Administration CAMI Library AAC 64D1, P.O. Box 25082, Oklahoma City, OK 73125 (1 Copy for Civil Aeromedical Institute)	1
Headquarters, U.S. Air Force Bolling AFB, DC 20332 (1 Copy for Office of the Surgeon General (SGP))	1